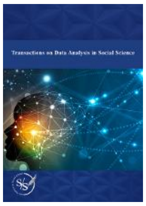
Transactions on Data Analysis in Social Science 1(3) (2019) 153–162

ISSN Online: 2821-1936

Transactions on Data Analysis in Social Science

Journal Homepage: https://transoscience.ir



Comparison of the Effect of 10 and 20 Grams of Creatine on GH, IGF-1 Levels in Professional Basketball Players after a Session of Intense Interval Training

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ARTICLE INFO

Article History:
Received 21 May 2019
Received in revised form 9 August 2019
Accepted 21 September 2019
Available online 27 September 2019

Keywords:
Creatine, Intense Interval Training, Growth Hormone, IGF-1 Hormone

ABSTRACT

The present study aimed to compare the effects of 10 and 20 grams of creatine supplementation on growth hormone (GH) and insulin-like growth factor-1 (IGF-1) levels in professional basketball players following a single session of high-intensity interval training. For this purpose, 21 professional basketball players were selected through convenience sampling and randomly assigned to three groups of seven participants each: 10-gram creatine loading, 20-gram creatine loading, and a placebo control group. Blood sampling was conducted at three stages: before the initiation of creatine or placebo supplementation; 24 hours after six days of creatine loading; and 24 hours after performing the high-intensity interval training session. Data were analyzed using repeated-measures analysis of variance (ANOVA). The results indicated that supplementation with either 10 or 20 grams of creatine had no significant effect on GH or IGF-1 levels in professional basketball players. However, high-intensity interval training had a significant effect on the levels of GH and IGF-1, leading to a marked increase in both hormones.

1. INTRODUCTION

High-intensity sports activities such as basketball rely heavily on rapid resynthesis of adenosine triphosphate (ATP). This regeneration is primarily mediated through the phosphagen, phosphocreatine–creatine, and glycolysis–glycogenolysis energy systems. These sources are continuously depleted and replenished during exercise. In intermittent activities involving short bursts of explosive power, rapid energy depletion and increased accumulation of lactic acid are among the main factors contributing to early fatigue and subsequent performance decline during the course of competitions [1].

To minimize this decline, many athletes use various ergogenic aids, including specialized training, nutritional strategies, and dietary supplements. It has been suggested that creatine supplementation, by increasing phosphocreatine stores, may enhance ATP resynthesis during very high-intensity activity [2] and thereby help delay the onset of fatigue. Numerous studies have emphasized the ergogenic value of creatine supplementation

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http://dx.doi.org/10.47176/TDASS.2019.153

over a period of 5 to 7 days in athletes' diets. In most cases, findings have shown that creatine content increases by 10–25%, while intramuscular phosphocreatine concentration rises by 20–40%, consequently accelerating ATP resynthesis during short-term, high-intensity exercise and improving performance in repeated bouts of maximal effort [3–4].

For example, Hoffman et al. (2005) administered a 6-gram creatine load for six days to 40 active men, who subsequently performed the Wingate test three times. Results indicated that creatine supplementation had a significant effect on peak power, mean power, and total work output [5]. Volek et al. (1997) examined the effects of consuming 25 grams of creatine for seven days and reported increased total work performed across five sets of bench press and squat exercises [6]. Snow et al. (2018) investigated the effects of creatine supplementation on sprint performance during repeated high-speed efforts and observed improvements in participants' sprint velocity [7]. Similarly, Kak and Caril (2013) examined the effects of six months of creatine supplementation on anaerobic capacity and muscle hypertrophy and found a significant reduction in blood lactate levels accompanied by increased anaerobic capacity and hypertrophy [8].

In recent years, extensive efforts have been made to elucidate the cellular and molecular mechanisms underlying skeletal muscle hypertrophy and atrophy. According to several researchers, growth hormone (GH) indirectly promotes tissue growth by stimulating the liver and, to a lesser extent, other tissues to produce a group of small proteins known as somatomedins, which exert strong anabolic effects on various tissues [9]. Many of the effects of somatomedins resemble those of insulin; therefore, they are collectively referred to as insulin-like growth factors (IGFs). Of the four known somatomedins, somatomedin C, or IGF-1, is the most important [10].

Insulin-like growth factor 1 (IGF-1) is a key member of the somatomedin family that plays a central role in growth, cell proliferation, differentiation, metabolism, and survival [11]. IGF-1 is a 70-amino-acid polypeptide that circulates in the plasma, largely bound to insulin-like growth factor binding proteins (IGFBPs). The liver is the primary source of circulating IGF-1. The hypothalamic nuclei, which are regulatory centers in the brain, control the secretion of growth hormone–releasing hormone (GHRH). These nuclei are activated through the neurotransmitters adrenaline and noradrenaline, leading to the release of GHRH into the hypophyseal portal system, where it stimulates the anterior pituitary to produce GH. GH then circulates to the liver and other peripheral tissues, where it promotes the synthesis of IGF-1 [12].

A unique characteristic of mature skeletal muscle is its intrinsic capacity to adapt to a wide range of physiological stimuli, such as various exercise modalities. When the muscle is subjected to overload, IGF-1 either independently or through GH stimulates myogenesis [13]; however, the precise mechanisms involved remain unclear. One of the main pathways for IGF-1 production involves GH-mediated stimulation of the liver, though other tissues such as muscle, brain, and bone can also synthesize IGF-1. Given its central role in metabolic regulation, research on this growth factor continues to expand [14]. Furthermore, the GH–IGF-1 axis has notable effects on bone growth and health, metabolic adaptation to both short- and long-term exercise, and overall organismal adjustment to physical activity [15].

Acute and short-term exercise serves as a potent physiological stimulus for the hypothalamus, promoting GH secretion and, consequently, the production of both GH and IGF-1 [16]. Suchy et al. (2004) examined the effects of intense exercise on GH and IGF-1 levels and found that a single bout of vigorous exercise significantly increased GH secretion, though it did not significantly affect IGF-1 concentration [17]. Hassani Ranjbar et al. (2012) reported that one session of intense resistance exercise elevated IGF-1 and its related hormones [18]. Other studies have attributed post-exercise increases in IGF-1 to its insulin-like metabolic effects rather than its hypertrophic effects, further complicating the interpretation of this hormone's role [19].

A review of the existing literature indicates that comparative studies investigating the effects of 10 and 20 grams of creatine on performance and anabolic hormones are limited. Most previous studies have included small sample sizes, lacked proper control over exercise conditions, and did not assess hormonal changes, thereby limiting the generalizability of their findings. In contrast, the present study specifically focused on participants of relatively homogeneous athletic level (professional basketball players) engaged in a single, well-defined sport. Moreover, the performance assessment employed a test relevant to basketball, and hormonal responses specifically GH and IGF-1 were measured alongside performance outcomes.

Accordingly, the main research questions of the present study are as follows:

Does loading with 10 and 20 grams of creatine following high-intensity interval training affect GH and IGF-1 levels in professional basketball players?

Is there a significant difference between the effects of 10-gram and 20-gram creatine doses on GH and IGF-1 levels following high-intensity interval training in professional basketball players?

2. RESEARCH METHODOLOGY

The present research is of a semi-experimental nature, employing a repeated-measures design that was conducted in the field. Additionally, considering the results, it is categorized as applied research. The research sample consisted of 21 professional male basketball players from Isfahan, aged between 19 and 30 years (Age: 27.19 ± 4.40 years, Weight: 28.88 ± 14.8 kilograms, BMI: 25.50 ± 2.47 kg/m²). The participants volunteered and cooperated with the study, and they were purposefully selected from the available population.

The participants were randomly assigned to three groups, each comprising 7 individuals: a group receiving 10 grams of creatine supplement, a group receiving 20 grams of creatine supplement, and a control group.

For data collection, the following tools were utilized:

1. Informed consent form
2. Medical and physical activity history questionnaire
3. Dietary recall questionnaire
4. Diagnostic Biochem Canada (dbc) kit, with a 2% coefficient of variation (cv) and 0.1-80 ng/ml sensitivity for measuring serum GH concentration
5. CLIA kit from Diasorin-USA, with a 6.2% cv and 3-1500 ng/ml sensitivity for measuring IGF-1 concentration
6. Creatine supplement sourced from the United States and approved by the Ministry of Health, provided in powder form by Poyan Company.

The research methodology involved both subjective and objective measures, including participant self-reports and biochemical analyses, to comprehensively assess the impact of creatine supplementation on GH and IGF-1 levels in professional basketball players after intense interval training**.**

3. IMPLEMENTATION METHOD

The present study employed a quasi-experimental design with repeated measures, conducted under field conditions. Based on its objectives, the research is categorized as applied in nature. The study sample consisted of 21 professional male basketball players from Isfahan, aged 19–30 years (mean age: 27.19 ± 4.40 years; body weight: 88.28 ± 8.14 kg; BMI: 25.50 ± 2.47 kg/m²), who were selected through convenience sampling and voluntarily agreed to participate in the study. Participants were randomly assigned to three groups of seven: 10 g creatine loading, 20 g creatine loading, and control (placebo).

2.1. Data Collection Instruments

The following instruments were used for data collection:

- Informed consent form
- Medical and physical activity history questionnaire
- Dietary recall questionnaire

- Diagnostic Biochem DBC (Canada) kit for serum GH analysis (inter-assay CV = 2%, sensitivity = 0.1–80 ng/ml)
- Diasorin-USA CLIA kit for IGF-1 measurement (inter-assay CV = 2.6%, sensitivity = 3–1500 ng/ml)
- Creatine supplement (powder form, manufactured in the USA, approved by the Iranian Ministry of Health, supplied by Pouyan Company)

2.2. Procedure

Before the start of the study, a briefing session was held to explain the research objectives and procedures. Participants were informed that they were taking part in a research project and that their cooperation would contribute to the successful completion of the study. Instructions on proper supplement consumption were also provided.

Blood sampling was scheduled for 8:00 a.m., and participants were required to be fasting at the time of sampling. Written informed consent was obtained from all participants, and a dietary recall questionnaire was distributed to document nutritional intake. After completing the initial dietary recall, participants were asked to maintain similar eating patterns during subsequent stages, with the questionnaire repeated 48 hours before each later sampling session to verify compliance.

Blood samples were collected at three stages:

1. **Baseline** – prior to creatine or placebo supplementation;
2. **Post-supplementation** – 24 hours after six consecutive days of creatine loading;
3. **Post-exercise** – 24 hours after performing the high-intensity interval training protocol.

At each stage, an 8 cc blood sample was drawn from the antecubital vein while the participant was seated. All samples were collected at 8 a.m. following 10 hours of overnight fasting. To prevent post-sampling hypotension, each participant received a small piece of cake and a glass of milk (approximately 300 kcal).

Labeled blood samples were stored at room temperature for one hour, then centrifuged using a Sahand centrifuge (Iran) at 2500 rpm for 30 minutes. The separated serum was then frozen at −18 °C for subsequent biochemical analysis.

For the high-intensity interval exercise, the Running-Based Anaerobic Sprint Test (RAST) protocol was employed, following the procedure described by Hamzezadeh Boroujeni et al. (2013) [20]. The RAST protocol consisted of six 35-meter sprints performed at maximal speed, with 10 seconds of rest between each sprint.

2.3. Statistical Analysis

Descriptive statistics were used to calculate measures of central tendency and dispersion. The Shapiro–Wilk test assessed data normality, and Levene’s test was used to examine the homogeneity of variances. For inferential analysis, a repeated-measures ANOVA was conducted to determine the effect of supplementation on the dependent variables. Additionally, a one-way ANOVA was used to compare the effects of different supplement doses. All analyses were performed using SPSS version 22 (IBM Corp., Armonk, NY, USA).

4. RESEARCH FINDINGS

Table 1 presents the mean and standard deviation of the dependent variables across different groups and experimental stages.

Table 1. Mean and standard deviation of the dependent variables across different groups

Group	Stage	GH (ng/ml)	IGF-1 (ng/ml)
10 g Creatine	Pre-test	11.85 ± 1.34	103.29 ± 3.09
	Post-test I	12.14 ± 1.34	105.71 ± 2.98
	Post-test II	14.57 ± 1.39	115.85 ± 4.67
20 g Creatine	Pre-test	11.28 ± 1.88	104.00 ± 3.16
	Post-test I	12.57 ± 2.07	106.14 ± 1.95
	Post-test II	16.28 ± 2.56	116.57 ± 3.50
Placebo	Pre-test	12.42 ± 1.13	102.00 ± 3.95
	Post-test I	12.71 ± 1.97	104.00 ± 2.70
	Post-test II	15.42 ± 2.14	114.57 ± 4.03

Table 2 presents the results of the within-group repeated measures ANOVA for GH and IGF-1 levels in each experimental group.

Table 2. Results of the Within-Group Repeated Measures ANOVA for GH and IGF-1 Levels in Each Group

Variable	Group	Sum of Squares	df	Mean Square	F	Sig.	η²
GH	10 g Creatine	31.143	2	15.571	9.909	0.003	0.623
	20 g Creatine	94.381	2	47.190	9.943	0.003	0.624
	Placebo	38.381	2	19.190	4.836	0.029	0.446
IGF-1	10 g Creatine	622.571	2	311.286	43.726	0.0001	0.879
	20 g Creatine	633.23	2	316.61	55.25	0.0001	0.902
	Placebo	638.857	2	319.429	29.22	0.0001	0.830

The results of the within-group repeated measures ANOVA on the measurement stages revealed that the consumption of 10 g of creatine after one session of high-intensity interval training had a significant effect on GH levels in professional basketball players ($F(2,12) = 9.909$, $p = 0.003$, $\eta^2 = 0.623$). However, the Bonferroni post hoc test indicated that creatine intake itself did not have a significant effect on GH levels ($p = 0.604$). A significant increase of 2.429 ng/ml in GH concentration was observed following the training session ($p = 0.024$).

Similarly, the consumption of 20 g of creatine after one session of high-intensity interval training showed a significant effect on GH levels ($F(2,12) = 9.943$, $p = 0.003$, $\eta^2 = 0.624$). According to the Bonferroni test, creatine intake alone did not produce a significant difference ($p = 0.263$); however, GH levels increased significantly by 3.714 ng/ml post-exercise ($p = 0.037$).

Furthermore, the placebo group also demonstrated a statistically significant change in GH levels following one session of high-intensity interval training ($F(2,12) = 4.836$, $p = 0.029$, $\eta^2 = 0.446$). Nonetheless, the Bonferroni post hoc analysis showed no significant effect of placebo consumption on GH ($p = 0.736$). Despite this, GH levels significantly increased by 2.714 ng/ml after exercise ($p = 0.047$).

As shown in Table 2, the within-group repeated measures ANOVA further indicated that consuming 10 g of creatine after one session of high-intensity interval training had a significant effect on IGF-1 levels in professional basketball players ($F(2,12) = 43.726$, $p = 0.001$, $\eta^2 = 0.879$). The Bonferroni post hoc test showed that creatine intake itself did not significantly affect IGF-1 levels ($p = 0.084$); however, a significant increase of 10.143 ng/ml in IGF-1 concentration was observed following exercise ($p < 0.0001$).

In addition, the 20 g creatine group exhibited a significant effect on IGF-1 levels after high-intensity interval training ($F(2,12) = 55.25$, $p < 0.0001$, $\eta^2 = 0.902$). Although creatine consumption did not significantly influence IGF-1 levels alone ($p = 0.078$), IGF-1 concentration significantly increased by 10.429 ng/ml after exercise ($p < 0.0001$).

Similarly, the placebo group demonstrated a significant effect on IGF-1 levels after a single session of high-intensity interval training ($F(2,12) = 29.22$, $p < 0.0001$, $\eta^2 = 0.830$). The Bonferroni analysis showed no significant difference due to placebo intake ($p = 0.058$), yet a significant increase of 10.571 ng/ml in IGF-1 levels was observed post-exercise ($p = 0.002$).

After analyzing within-group differences, between-group differences were examined using one-way ANOVA for each measurement stage.

Table 3. Results of the One-Way ANOVA for Changes in GH and IGF-1 Across Each Measurement Stage

Variable	Measurement Stage	Sum of Squares	df	Mean Square	F	Sig.	η^2
GH	Pre-test	4.571	2	2.286	1.029	0.378	0.103
	After Supplementation	1.238	2	0.619	0.186	0.832	0.020
	After Exercise	10.286	2	5.143	1.174	0.332	0.115
IGF-1	Pre-test	14.381	2	7.190	0.612	0.553	0.064
	After Supplementation	18.000	2	9.000	1.347	0.285	0.130
	After Exercise	14.381	2	7.190	0.428	0.658	0.045

As shown in Table 3, no statistically significant differences were observed between groups in GH levels at any stage of measurement namely, the pre-test ($F(2,18) = 1.029$, $p = 0.378$, $\eta^2 = 0.103$), after supplementation ($F(2,18) = 0.186$, $p = 0.832$, $\eta^2 = 0.020$), and after exercise ($F(2,18) = 1.174$, $p = 0.332$, $\eta^2 = 0.115$).

Similarly, no significant between-group differences were found in IGF-1 levels across all stages of measurement. These included the pre-test ($F(2,18) = 0.612$, $p = 0.553$, $\eta^2 = 0.064$), after supplementation ($F(2,18) = 1.347$, $p = 0.285$, $\eta^2 = 0.130$), and after exercise ($F(2,18) = 0.428$, $p = 0.658$, $\eta^2 = 0.045$).

5. DISCUSSION AND CONCLUSION

The present study aimed to compare the effects of 10 g and 20 g of creatine supplementation on serum GH and IGF-1 levels in professional basketball players following a single session of high-intensity interval training. The findings revealed that creatine supplementation combined with intense interval exercise increased GH levels; however, no statistically significant differences were observed among the creatine supplementation groups and the placebo group. The results suggest that the significant increase in GH observed in the placebo group was primarily due to the effects of high-intensity interval exercise rather than the different doses of creatine. This finding is consistent with the results reported by Sheikholeslami Vatani et al. (2009), who also found no significant effect of creatine supplementation on GH secretion [21]. Similarly, Quesada and Gillan (2013) reported that creatine supplementation did not affect GH levels [22]. Moreover, Gallant et al. (2015) examined the effects of 2 to 6 days of creatine supplementation on GH levels in 31 men and 30 women and found that although body weight increased in both groups, GH concentrations remained unaffected [23]. The study by Smith et al. (1998) also aligns with the present findings. Smith and colleagues (1998) investigated the effects of a 5-day creatine supplementation protocol on GH levels in college women [24]. Fifteen participants were randomly assigned to creatine and placebo groups, receiving 5 g of creatine four times per day (20 g/day). Their results showed that although the creatine group exhibited a significant increase in body weight compared with the placebo group, GH levels did not significantly change.

Overall, it appears that various factors including experimental conditions, type and intensity of the training protocol, duration, volume, participants' skill level, and timing of blood sampling may have contributed to the diversity of results across studies. In other words, the combined effect of the training protocol and creatine supplementation in the current study did not provide sufficient stimulus or potential to enhance GH secretion. Moreover, it is possible that GH concentrations increased at later time intervals following exercise, which were not measured due to practical limitations of the present study.

The findings also indicated that resting GH concentrations significantly increased after high-intensity interval training. Resting GH levels likely reflect the physiological state of the muscle tissue, and changes in these levels may occur during different training phases as intensity and volume fluctuate. Since GH was used in this study as an indicator of the anabolic–catabolic balance in skeletal muscle, the observed increase in resting GH suggests the activation of anabolic mechanisms induced by training. Although the precise intracellular signaling and mechanism of GH action remain unclear, GH may enhance muscle protein synthesis through both IGF-1–dependent (Veloso, 2008; Nielsen et al., 2008; Asbjornsen et al., 2009) [25–27] and IGF-1–independent (Liu et al., 2003; Asbjornsen et al., 2009) [27–28] pathways. Considering that GH stimulates IGF-1 production in tissues, it could be expected that elevated GH levels during such programs would subsequently lead to greater IGF-1 synthesis. However, the absence of a noticeable increase in IGF-1 immediately after intense exercise may be due to the time required for GH to trigger IGF-1 production in tissues [29].

The results of this study also demonstrated that creatine supplementation combined with high-intensity interval exercise increased IGF-1 levels; nevertheless, no significant differences were observed among the creatine and placebo groups. This again suggests that the increase in IGF-1 resulted primarily from the intense exercise protocol rather than the creatine doses. These findings are consistent with those of Gallant et al. (2015), who found that although body weight increased following short-term creatine supplementation (2–6 days), no significant changes occurred in anabolic hormone levels [23]. Likewise, Smith et al. (1998) reported that a 5-day creatine supplementation regimen in female college students did not significantly affect IGF-1 levels despite a significant increase in body weight [24].

The present study further showed that resting IGF-1 levels significantly increased following high-intensity interval exercise. However, it should be noted that potential increases in intramuscular IGF-1 production, which may not be reflected in serum measurements, were not examined due to methodological limitations this could partly explain the observed rise in resting IGF-1. Previous research has indicated that the peak concentration of IGF-1 occurs 16 to 28 hours after GH stimulation, implying that the increase in IGF-1 levels between 3 and 28 hours post-exercise may result from the delayed response to elevated GH levels [30–31]. Therefore, following high-intensity interval exercise, GH levels increase substantially, and a subsequent rise in IGF-1 is expected several hours later [29]. GH not only stimulates IGF-1 production in muscle tissue but also activates IGF-1 receptors [25–26].

Based on the results of the present study, it can be concluded that short-term creatine supplementation at doses of 10 g and 20 g does not significantly enhance anabolic hormone responses in professional athletes when combined with high-intensity interval exercise. It appears that creatine, at least in the short term, does not influence the secretion of anabolic hormones such as GH and IGF-1 under these conditions. Given the significant stimulatory effect of high-intensity interval training itself on anabolic hormones in professional basketball players, it is recommended that coaches and athletes incorporate such training protocols particularly when time is limited to optimize anabolic hormonal responses and performance adaptations.

Transparency Statement

The data supporting this study are available upon reasonable request to the corresponding author, subject to ethical and confidentiality considerations.

Acknowledgments

We would like to express our gratitude to all individuals who contributed to this project.

Declaration of Interest

The authors declare that they have no competing interests.

Funding

This research received no specific grant from any funding agency, commercial, or not-for-profit sectors.

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